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Re: Comments on the Baseline Human Health Risk Assessment (BHHRA), Feasibility Study Report (FSR), and Preferred Alternative for the Vasquez Boulevard/Interstate 70 (VB/I70) Superfund Site

The comments in this memorandum are subdivided into 3 main parts. The first two parts address the methods and approach used by EPA in its assessment and presentation of the health risk posed by arsenic and lead, respectively, at the VB/I70 Superfund Site. The methods and approach in question were presented primarily in the Baseline Human Health Risk Assessment, and the comments contained herein supplement those submitted previously during the comment period on the draft of the Baseline Risk Assessment. The third part of this memorandum provides comments on the Feasibility Study Report, and the preferred remedial alternative for VBI70 proposed by EPA in May, 2002.

A. Comments regarding the methods and analytical approach used by EPA to assess health risks from arsenic.

A.1. As was noted in prior comments on the Draft Baseline Human Health Risk Assessment, the slope factor used by EPA in assessment of cancer risk from ingestion of arsenic in soils continues to be the value of 1.5 mg/kg/day contained in the IRIS database. This value, which has remained unchanged in the IRIS database since 1988, is based solely on the risk of arsenic-induced skin cancer. As discussed in detail in two recent reports by the National Research Council (NRC 1999, 2001), and as acknowledged by U.S. EPA itself in its adoption last year of a revised MCL for arsenic in drinking water (EPA, 2001), there is extensive scientific data that establishes that arsenic ingestion

increases the risk of lung cancer and bladder cancer. The analysis presented in the most recent NRC report, (NRC, 2001) indicates that the slope factor (i.e. cancer risk divided by arsenic dose in mg/kg-day) associated with arsenic-induced lung and bladder cancer combined is likely to exceed 1.5 mg/kg-day by a considerable margin. NRC (2001) concluded that the combined excess lung and bladder cancer risk associated with a drinking water arsenic concentration of 10 µg/L (0.010 mg/L) was likely to be equal to *or greater than* 1 in 1,000 (one in one thousand). In this same report, NRC noted and utilized recent findings that indicate that a typical 70 kg adult consumes 1 liter of tap water per day. The NRC analysis can be used to calculate a slope factor as follows:

slope factor = excess risk/(mg/kg-day) = 0.001/(0.01mg/L x l L/70 kg) = 7.0 mg/kg-d

Accordingly, the discussion on "Cancer Effects" on pages 65 and 66 of the Baseline Human Health Risk Assessment should be revised to reflect the fact that the most recent NRC assessment supports the use of a slope factor of 7.0, a value nearly 5 times higher than the value of 1.5 mg/kg-d that was actually used in the cancer risk calculations. The implications of this higher slope factor for assessment of cancer risk posed by arsenic in soil at VB/I70 properties should be qualitatively and quantitatively addressed in a revision of the document, and in a revision of the preliminary action levels for arsenic set forth in EPA's risk management memorandum of October 19, 2001 (Appendix C of the Feasibility Study Report). [1] Carefully and explicitly addressing the implications of this recent NRC report would be consistent with an approach used elsewhere in the document of citing and incorporating calculations based on several recent studies or techniques, many of which have served to reduce risk estimates.

A.2. In the section on Acute Noncancer Effects of arsenic, the BHHRA (page 64) states that EPA, in a report written by Dr. Robert Benson, has established an acute RfD for arsenic of 0.015 mg/kg-d. This RfD was used in the identification of a soil exposure point concentration (EPC) of 47 ppm as a Preliminary Action

[1] Several other aspects of the discussion of cancer risk in the Baseline Human Health Risk Assessment appear outdated and merit revision. On page 65, rather than focus on skin cancer, the narrative should emphasize that arsenic causes lung cancer and bladder cancer, which are much more likely to be associated with fatal outcomes. It may be noted that in contrast to the cited paper by Morales et al (2000), NRC (2001) expressed a strong preference for the use of an external reference population in risk assessments using the SW Taiwanese dataset. The discussion on page 85 in the subsection entitled, "Uncertainty in Toxicity Factors" suggests that in vivo methylation may be a detoxification mechanism for arsenic, when the weight of recent evidence suggests otherwise (NRC, 2001). The discussion on page 85 may also be interpreted by some readers to infer that nutritional factors may exert a considerable influence on susceptibility to arsenic induced cancer; however, current evidence for such a hypothesis is scant (NRC, 2001)

Level based on acute hazard for a child with pica behavior (FSR, page 23). The narrative of the BHHRA states that the acute RfD of 0.015 mg/kg-d was derived from a study of individuals in India chronically exposed to arsenic in drinking water (Mazumder et al, 1998) in which 0.015 mg/kg-d was identified as a NOAEL for chronic skin lesions. No uncertainty factor was applied to the NOAEL derived from this study. Several concerns exist regarding this determination. First, the study in question was a survey of a chronic health endpoint, i.e. arsenic skin lesions, and was not designed to detect acute adverse health effects of arsenic. Second, the dose calculations used in that study must be interpreted with caution, because the study used an unspecified technique to "estimate daily water intake" and the actual data on water intake were not reported. Third, the study was cross-sectional in nature, and although it may be presumed that many of the subjects had longterm exposure to well-water, the length of time associated with water of a particular arsenic content was not specified. Fourth, skin lesions in that study were detected in some subjects, including one child under nine years of age, whose current arsenic dose was estimated to be less than 0.015 mg/kg-d.

As noted in the BHHRA, ATSDR has relied on alternative studies to derive an acute minimum risk level (akin to an acute RfD) of 0.005 mg/kg-d. It should also be noted that a recent FIFRA Scientific Advisory Panel (EPA, 2001) convened by EPA recommended that a margin of exposure ranging from 10 to 30 be applied to a LOAEL of 0.05 mg/kg for purposes of a short-term oral arsenic exposure guideline for children. This in effect identified an acute RfD of 0.005 to 0.0017 mg/kg-d. (I served as a member of this SAP, and endorsed the value of 0.005 mg/kg-d).

There is no dispute that major uncertainties exist regarding the assessment of acute arsenic risk posed by soil pica behavior, and that it also poses a considerable challenge with respect to risk management. The issue of acute RfD aside, major factors in the uncertainty of the risk assessment pertain to the frequency of pica behavior, the intake rate, and the absorption fraction associated with high dose ingestions of a soil matrix. In light of the human data available for determination of an acute LOAEL for arsenic, and EPA's traditional approach for assigning margins of exposure, the agency should discuss why it did not give at least equal consideration to selecting an acute RfD of 0.005 mg/kg-d (as opposed to 0.015 mg/kg-d) in setting the preliminary action level at VBI70.

B. Comments regarding the methods and analytical approach used by EPA to assess health risks from lead

B.1. In assessing the risk to young children of oral lead exposure from soil, the BHHRA noted that EPA, acting on policy established in the early 1990's, has identified 10 µg/dL as the "blood lead level at which effects that warrant avoidance begin to occur". In like manner, it noted that since 1994, EPA has "set as a goal that there should be no more than 5% chance that any child will have a blood lead value above 10 µg/dL" (BHHRA, page 89). However, in the section of the BHHRA that discusses the uncertainty in the lead hazard risk assessment, no mention is made of recent data that indicates that the level of 10 µg/dL may not be sufficiently protective. The history of public health recognition of the adverse effect of lead on children has been characterized by a progressive lowering of the blood lead level of concern over time. This fact was acknowledged by the US Centers for Disease Control and Prevention when it identified 10 µg/dL as the level of concern in its last major review of this topic in 1991. A recent study by Lanphear et al (2000) found that blood lead levels less than 5 µg/dL were associated with adverse neurocognitive outcomes in young children. The authors concluded, "Collectively, the results of the present analyses and other studies argue for a reduction in blood lead levels that are considered "acceptable" - from 10 µg/dL to 5 µg/dL or lower." The BHHRA and the FSR should discuss the implications for lead risk assessment and risk management, respectively, of the very real possibility that a reevaluation of this topic by the CDC in the near future may lower the blood lead level of concern to 5 µg/dL, or lower.

C. Comments regarding the Feasibility Study Report and EPA's Preferred Alternative for VBI70

C.1. The remedial action objective set forth for lead in soil is to "Limit exposure to lead in soil such that no more than 5 percent of young children (72 months or younger) who live within the VB/I70 site are at risk for blood lead levels higher than 10 µg/dL from such exposure." (FSR page 22).

Implementation of a successful strategy to achieve that goal, and verification of such success, requires that EPA clarify the manner in which it will consider in its analysis and plan the likelihood that children in the VBI70 study area have an elevated baseline in blood lead concentration from non-soil sources such as lead paint. The premise that there is an elevated background in blood lead within VBI70 is supported by the following: a) The socioeconomic demographics of the community, i.e. an urban community with a high proportion of Hispanic and African-American families, are associated with increases in

blood lead concentration relative to the national average (NCEH/CDC, 1997); b) the community has a high percentage of pre-1970 housing, a risk factor for lead paint exposure; c) the results of recent blood lead monitoring in the community collected by CDPHE are consistent with an elevation relative to national data (see BHHRA pp 103-104); d) a recent door to door survey of a Denver neighborhood with relatively similar demographic characteristics (Denver Childhood Blood Lead Survey, Final Report- January, 1996, CDPHE, 1996), found that 16.2% of children aged 12 to 35 months of age had blood lead concentrations in excess of 10 µg/dL; and e) 8 of 86 children (nearly 10%) screened by CDPHE in the VBI70 area on September 25, 2000 had blood lead concentrations greater than 10 µg/dL (see ATSDR Public Health Assessment, 2002, page 47).

A decision by EPA to acknowledge and incorporate this likely elevation in baseline blood lead concentration in its approach to limit the capacity of soil lead exposure to cause more than 5% of children to have blood lead concentrations in excess of 10 µg/dL may require not only a vigorous program of community education on lead hazard risk reduction, but also more stringent reductions in the acceptable concentration of lead in soil, and/or a program that will directly mitigate or eliminate non-soil sources of lead, particularly lead paint. There is authority and precedent within EPA and the Superfund program to consider these latter approaches. In this regard, it can be noted that there has been a growing trend within EPA in support of risk assessments that explicitly consider a community's *cumulative* exposure to toxicants such as lead in the design and implementation of a remedy. This has been discussed in two recent agency documents: 1) Guidance on Cumulative Risk Assessment. Part 1. Planning and Scoping (EPA, 1997); and 2) Framework for Cumulative Risk Assessment (EPA, 2002 draft). This latter document, currently an external review draft developed by EPA's Risk Assessment Forum, notes the following:

> One of the concepts that can be used in risk assessments (both for human health and ecological assessments) is that of vulnerability of the population or ecosystem. Vulnerability of a population places them at increased risk of adverse effect, and may be an important factor in deciding which stressors are important in doing a cumulative risk assessment. The Agency 's risk characterization policy and guidance (US EPA, 2000c) touches on this concept by recommending that risk assessments "address or provide descriptions of [risk to ] ...important subgroups of the population, such as highly exposed or highly susceptible groups ". Further, the Agency 's guidance on planning and scoping for cumulative risk assessments (US EPA, 1995b) recognizes the importance of "defining the characteristics of the population at risk, which include individuals or sensitive subgroups which may be highly susceptible to risks from stressors or groups of stressors due to their age,

> gender, disease history, size or developmental stage". That guidance also recognizes the potential importance of other social, economic, behavioral or psychological stressors that may contribute to adverse health effects (e.g., existing health condition, anxiety, nutritional status, crime and congestion). These same concepts may also be discussed as a group in terms of "population vulnerability."... The various ways in which a population may be vulnerable are discussed below in four categories: susceptibility, differential exposure, differential preparedness, and differential ability to recover....The second category of vulnerability is differential exposure. While it is obvious by examining a dose-response curve that two individuals at different exposure levels may have a different likelihood of effects, *this also extends to differences in historical exposure, body burden, and background exposure, which are sometimes overlooked in an assessment.* [emphasis added]. (EPA, 2002).

EPA is strongly urged to revise the BHHRA, the FSR, and its conception of the preferred alternative to provide a discussion of the socioeconomic demographics and elevation in non-soil lead exposure and body burden that likely characterize the VBI70 study area. EPA should indicate how it will consider cumulative lead exposure in devising, implementing, and verifying the effectiveness of the remedy. It should be noted that consideration of cumulative exposures is a recognized component of EPA's Environmental Justice initiative. Mr. Martin Halper of EPA's Office of Environmental Justice made a presentation on the significance of the Framework for Cumulative Risk Assessment document for environmental justice at the December, 2001 meeting of EPA's National Environmental Justice Advisory Council. According to the official meeting summary, Mr. Halper "stated that the framework document, which includes traditional quantitative considerations, as well as qualitative considerations, has the potential to affect the way in which EPA and other federal agencies operate." (EPA, 2001).

A recent EPA funded research report issued by the Environmental Law Institute suggested that, in the interest of environmental justice, EPA has statutory authority under CERCLA to directly address the hazards posed by lead based paint. The report stated:

> Section 104(a)(4) establishes exceptions to the limitations on EPA's removal and remedial authority that are contained in Section 104(a)(3). The limitations prevent EPA from taking removal or remedial action in response to releases or threats of releases from a naturally occurring substance from a location where it is naturally found; from products that are part of the structure of, and result in exposure within, residential buildings or business or community structures; or releases into public or

> private drinking water supplies due to deterioration of the system through ordinary use. Despite these limitations, Section 104(a)(4) allows EPA to respond to these types of releases or threats of releases of hazardous substances when it constitutes a "public health or environmental emergency" and no other person with authority and capability to respond will do so in a timely manner. 42 U.S.C. § 9604(a)(4). EPA has issued regulations implementing these provisions. 40 C.F.R. § 300.400(b). EPA has rarely used these exceptions to the limitations on its removal and remedial authority. EPA could, however, rely on this section to address hazardous substance releases in low-income communities and communities of color that may otherwise go unaddressed. This may include releases from products, such as asbestos or lead paint, that are part of the structure of buildings. They may also include releases into public or private drinking water supplies due to deterioration of the system through ordinary use, particularly in communities with limited financial resources for maintaining buildings and water systems. In addition, such releases may pose particular public health threats in many low-income communities and communities of color because of factors such as sensitive populations and cumulative risks. Furthermore, because many low-income communities and communities of color have limited resources, it may be likely that there are no other authorities with capability to respond to the releases. (Environmental Law Institute, 2001, page 151).

Members of EPA's Region VIII Environmental Justice team have participated in the VBI70 process to foster community involvement, but it is not clear how environmental justice concerns were incorporated in the FSR or EPA's development of a preferred alternative for VBI70. EPA should revise the FSR and its presentation of a preferred alternative to explicitly discuss how environmental justice concerns have been factored into design and selection of the remedy. In accordance with the above cited Environmental Law Institute Report, EPA should analyze whether existing mechanisms for detection and abatement of lead based paint within the VBI70 community have adequate scope and funding to reduce, in a timely fashion, the vulnerability of the community's children to this component of cumulative lead exposure. EPA should examine whether direct EPA support for lead paint abatement is warranted to help EPA achieve, in what may be a cost effective manner, a RAO for lead that incorporates the impact of cumulative lead exposure. [2]

[2] Consonant with the approach of considering cumulative exposure and environmental justice issues, the FSR and the process of selecting a remedial action objective for arsenic should examine the implications of the recent cancer study by CDPHE (2001) that adults within the VBI70 community may have increased exposure or vulnerability to other lung carcinogens. The Standardized Incidence Ratio (SIR) for lung cancer (both sexes) in a study area that encompassed the VBI70 community was 1.25 (95% C.I. 1.05 - 1.48). Because lung cancer is a major cause of mortality, an increase in SIR of this magnitude has considerable [footnote continued on next page]

C.2. In a memorandum to the Administrative Record File dated October 19, 2001, (FSR Appendix C, page 11), EPA identified 540 ppm as a preliminary action level for lead in soil requiring engineering (e.g. removal) action. The memorandum stated, "This is the soil concentration at the higher end of the range of soil concentrations that the IEUBK model predicts EPA's health goal will be exceeded." The parameter values resulting in derivation of this value were not specified, but based on Table 2 of the memorandum, it appears that 540 ppm may have been derived using default dietary lead values and a geometric standard deviation (GSD) of 1.2 µg/dL for blood lead concentration. If that were the case, the GSD value of 1.2 represents a departure from the default GSD value of 1.6. Per table 2, the default GSD value of 1.6 would yield a preliminary action level for lead in soil of 208 ppm. The BHHRA (page 101) provides a qualitative explanation of reasons why the default GSD value of 1.6 may overestimate the true GSD. It also provides the results of a an ISE model iteration that yielded a GSD of 1.2. [3] However, justification for the selection of a GSD value of 1.2 would be enhanced if EPA could provide a statistical analysis of the parameters used in the IEUBK that reveals that the overestimation inherent in the default value of 1.6 quantitatively supports a revised value of 1.2.

C.3. EPA's bulletin of May, 2002 identifying Clean-up Alternative 4 as the preferred alternative indicates that 306 properties require soil removal because of arsenic. Can EPA report how many of these properties require soil removal because of the cancer risk from RME soil exposure alone, and how many because of the combined cancer risk of RME soil exposure plus CTE garden vegetable consumption?

C.4. EPA's preferred alternative (Clean-up Alternative 4) contains as a key remedial component a Community Health Plan (CHP) intended to contribute to the implementation and verification of the remedial action objectives for lead and arsenic. The CHP intends to achieve this through a program of health education and biomonitoring. The goals of the CHP are laudable, and a CHP may have the capacity to improve public health within the VBI70 study area. However, in its present form, the information provided in the FSR is insufficient

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public health significance. It should also be noted that Hispanic and African-American children appear more likely than non-Hispanic white children to suffer from iron deficiency, a condition that may be at least additive with lead poisoning in having adverse impacts on neurocognitive development (CDC, 1998; CDC, 2002).

[3] It should be noted that the GSD value of 1.2 reported for the ISE model was derived using an age range for childhood exposure of 1 to 84 months (BHHRA, page 101). This appears to be slightly inconsistent with the RAO for lead in soil stated on page 22 of the FSR, which cites an age range of less than 72 months. The potential impact of this discrepancy, though possibly slight, should be explored.

to establish that the CHP will adequately satisfy several of the relevant primary balancing criteria required for selection of a remedial alternative.

C.4.a. Although the FSR noted that there is no precedent that establishes the efficacy of health education in reducing soil pica behavior, it cited examples of parental education programs dealing with childhood depression and drug use as evidence that an educational intervention will be effective. This analysis fails to consider that soil pica behavior in toddlers may be an innate behavior that is not amenable to substantive reduction through education. Can EPA point to evidence that counters the opinion of David Mellard, PhD of ATSDR in a letter to Bonnie Lavelle of EPA dated June 19, 2001, in which he stated, "Soil-pica behavior is an innate behavior in 1 and 2 year old children and teaching them about the hazards of such behavior will not stop that behavior. While it is possible to educate parents about the hazards of soil-pica behavior, it is not reasonable to assume that parents can watch their children constantly to prevent that behavior. ATSDR views health education on soil-pica behavior as an interim measure to reduce the risk from soil-pica behavior while more permanent solutions are investigated."

C.4.b. Without providing logistical details or quantitative estimates, the FSR states that a voluntary childhood biomonitoring program will achieve a sufficient participation rate to provide detection and secondary prevention of elevated exposure to lead and arsenic. Can EPA examine and comment on whether the rate of participation in the nearby Globeville biomonitoring program provides confidence that a somewhat similar program for VBI70 will achieve an acceptable participation rate? At moderate dose levels, the half-time of arsenic excretion via the urine is a matter of a few days to a week. After estimating the frequency of soil pica behavior among the community's approximately 2500 young children, and the anticipated biomonitoring participation rate, can EPA present a statistical power analysis that examines the feasibility of a urine arsenic biomonitoring program for detecting, with an acceptable degree of confidence, the true prevalence or incidence of elevated arsenic exposure from soil-pica behavior? What criteria would EPA apply to assess whether health education was an acceptable remedy for reduction of soil pica behavior?

In like manner, can EPA explain how it proposes to utilize the results of the blood lead monitoring program to assess the effectiveness of the CHP in meeting the RAO for lead? The lack of clarity regarding the scope of the RAO for lead with respect to soil-related versus cumulative lead exposure was noted above. If EPA will consider the RAO for lead to be achieved by a specified change in the contribution of soil lead exposure to the percentage of children with blood lead concentrations above 10 µg/dL, what criteria will it employ in this assessment? In the event of case management investigations for specific children with elevated blood lead levels, how will the relative contribution of

exposure to lead in soil and paint be determined, particularly when lead is present in both media? If EPA will determine that the RAO for lead is achieved when less than 5% of children in VBI70 have blood lead concentrations less than 10 µg/dL due to all (i.e. cumulative) lead sources, what level of participation in the biomonitoring program will be necessary to detect this level of success with confidence?

C.4.c. The FSR states that the CHP will be a factor in establishing the "long-term effectiveness and permanence" of the preferred Clean-up Alternative. By its very nature, it would appear that the effectiveness of health education and secondary prevention through biomonitoring will persist only as long as the CHP remains active. However, if the detection of sources of hazardous lead exposure through the CHP results in their eventual abatement, then the CHP may be regarded as having contributed to permanent effectiveness at those particular properties. By what criteria will EPA judge the CHP to have successfully contributed to a permanent remedy that persists after the CHP is discontinued? [4]

C.4.d. The FSR states that the CHP will be readily implementable, due in part to the existence of organizational structures for lead poisoning detection and prevention at the state and local levels. To what extent will the effectiveness of the CHP developed by EPA be dependent on the continued existence of these state and local programs? If such dependence is significant, will EPA provide funding, above and beyond that envisioned for the VBI70 CHP alone, to assure the longterm stability and existence of the state and local lead hazard reduction programs?

C.5.d. Notwithstanding the lack of adequate details on the CHP within the narrative portion of the FSR, the budget for the CHP presented in Appendix B, Tables B-7 and B-8, suggests that the scope of the program will be insufficient to accomplish the intended goals. For example, the budget suggests that approximately one half of an FTE (full time equivalent, or full-time position) will

[4] It is noteworthy that a recent research report by the Environmental Law Institute observed that establishment of truly permanent solutions is a component of environmental justice. The authors wrote "The CERCLA cleanup provisions state a strong preference for cleanups that are permanently protective of public health. This preference, along with other stated goals, is consistent with ensuring that protective remedies are selected for sites in communities of color and low-income communities. Therefore, EPA should be able to consider environmental justice factors in developing and implementing remedy selection procedures. In addition to the general authority granted under this section, the statute specifically requires EPA to take into account in selecting among alternative remedies "the propensity to bioaccumulate" of hazardous substances. See 42 U.S.C. § 9621(b)(1)(C). The statute also attempts to hold EPA accountable in circumstances in which it does not select permanent treatment remedies by requiring an explanation. This provision, in particular, could benefit communities of color if used proactively, in light of studies that have indicated that EPA is more likely to select non-treatment remedies for sites in communities of color than for sites in white communities. See Ferris at 673 (citing Lavelle & Coyle)." [Environmental Law Institute, 2001, page 160].

be sufficient, on an annual basis, to publicize the program, and obtain biological monitoring samples on 700 children. This is derived from Table B-8, which allocates 268 person hours to Education/Public Awareness, and 800 hours (400 hours x 2) for collection of urine arsenic and blood lead samples. This subtotal, 268 + 800 = 1068, represents approximately one person working slightly more than half time for a year. A total of only 400 additional hours, or approximately one-fifth of a full time position, is envisioned for case management services. Thus, the FSR appears to suggest that the key components of an effective CHP, i.e. publicity, recruitment, sampling, and case management, can be accomplished by less than one full time position. This seems doubtful, particularly in a community where a relatively high proportion of children may have elevations in blood lead. The section of the budget dealing with "source investigation and remediation" indicates that an average of 33 residences, or less than one percent of the area residences, will be investigated each year. EPA should present a relatively detailed narrative that explains how the seemingly modest level of subject recruitment, case management, and residential investigations set forth in the budget will constitute a CHP sufficient to assure that the public health needs of the community are addressed.

C.6. The University of Colorado Health Sciences Center is currently (summer, 2002) conducting an investigation, funded by EPA and ATSDR, that will gather information on childhood soil contact, and arsenic and lead exposure, in the VBI70 study area. It seems likely that the information gathered in this study will contribute to a greater understanding of the risks posed by arsenic and lead exposure in the study area, as well as the capacity of a biomonitoring program to effectively assess the situation. This information may also assist in the development of an optimal remedy, and provide information on the required scope and resources needed for a community health plan.

In light of 1) the questions and concerns expressed in this memorandum regarding selected aspects of the health risk assessment and the uncertainty analysis in the BHHRA, 2) the data-gaps in the discussion of remedies in the FSR, and 3) the impending availability of information from the summer health study, it is respectfully requested that the comment period for the VBI70 docket remain open until the revised or supplemental information has been provided and reviewed.

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